

# STARS

University of Central Florida  
**STARS**

---

HIM 1990-2015

---

2013

## Examination of the relationship between sport concussion and long term neurodegenerative and psychological disorders: a literature review

Vivian Rivera  
*University of Central Florida*



Part of the [Psychology Commons](#), and the [Sports Sciences Commons](#)

Find similar works at: <https://stars.library.ucf.edu/honorstheses1990-2015>

University of Central Florida Libraries <http://library.ucf.edu>

This Open Access is brought to you for free and open access by STARS. It has been accepted for inclusion in HIM 1990-2015 by an authorized administrator of STARS. For more information, please contact [STARS@ucf.edu](mailto:STARS@ucf.edu).

---

### Recommended Citation

Rivera, Vivian, "Examination of the relationship between sport concussion and long term neurodegenerative and psychological disorders: a literature review" (2013). *HIM 1990-2015*. 1456.  
<https://stars.library.ucf.edu/honorstheses1990-2015/1456>



EXAMINATION OF THE RELATIONSHIP BETWEEN SPORT  
CONCUSSION AND LONG TERM NEURODEGENERATIVE AND  
PSYCHOLOGICAL DISORDERS: A LITERATURE REVIEW.

by

VIVIAN R. RIVERA

A thesis submitted in partial fulfillment of the requirements  
for the Honors in the Major Program in Athletic Training  
in the College of Health and Public Affairs  
and in The Burnett Honors College  
at the University of Central Florida  
Orlando, Florida

Spring Term 2013

Thesis Chair: Dr. Kristen Schellhase

© 2013 Vivian R. Rivera

## **ABSTRACT**

**Background:** According to the Center for Disease Control and Prevention, approximately 1.6 to 3.8 million Americans suffer a sports related concussion each year. Concussion is defined as a transient alteration of the brain structure caused by a direct or indirect force. During the last decade, a vast amount of clinical research on the long term effects of repetitive head trauma has occurred, especially on the subject of chronic traumatic encephalopathy (CTE), depression and dementia.

**Objective:** The purpose of this literature review is to examine the literature pertaining to multiple concussion and the long-term effects of multiple concussion such as neurodegenerative diseases and psychological.

**Methods:** A literature review was conducted using an electronic search of the following databases: MEDLINE, Cochrane Database of Systematic Reviews, and SportDiscus. The key search terms included were concussion, “sport concussion” and “sports concussion”. One of the above three terms needed to be in conjunction with one of the following key search terms: depression, dementia, "mild cognitive impairment", “chronic traumatic encephalopathy” (CTE), or “psychological disorder”. Additional inclusion criteria also included studies that

targeted the adult athlete population who had sustained more than one concussion. Studies only were included if they were peer-reviewed, in the English language, and were published after 1990. To be included in the review, the study must have examined the long term effects of repetitive concussion.

**Results:** The research completed to date suggests there is a strong correlation between the number of concussions an athlete suffers and the long-term ramifications of neurodegenerative and psychological disorders. However, more research is needed.

**Keywords:** concussions, chronic traumatic encephalopathy, dementia, depression

## **DEDICATIONS**

To my parents and grandparents: Thank you for all the support, love and for always believing in me.

To my committee: Dr. Schellhase, Jennifer Plant, and Dr. Pabian, thank you for all the support throughout this semester, without you this wouldn't be possible.

To my friends: Thank you for motivating me throughout this process.

## **ACKNOWLEDGMENT**

I would like to express my gratitude to my committee member, Dr. Patrick Pabian who dedicated his time and knowledge to guide me through the course of writing this thesis.

I would also like to give my gratitude to my committee member, Jennifer Plant, for always knowing what to say and keeping my spirits high. Special thanks to my thesis chair, Dr. Kristen Schellhase, for all the support and words of encouragement throughout this program. To all of my friends, thank you for everything you have done for me over these past four years at the University of Central Florida. Lastly, thank you my parents for guiding me through the right path in life and making me the woman I am today. I love you.

## TABLE OF CONTENTS

CHAPTER ONE: INTRODUCTION .....	1
CHAPTER TWO: METHODS.....	3
CHAPTER THREE: LITERATURE REVIEW .....	4
CONCUSSION.....	4
CTE.....	9
DEPRESSION .....	13
MILD COGNITIVE IMPAIRMENT (DEMENTIA) .....	14
CHAPTER FOUR: DISCUSSION.....	17
THE ROLES OF THE ATHLETIC TRAINER.....	17
First Provider of Care.....	17
Educators .....	19
Advocates .....	20
Limitations and Further Research.....	21
APPENDIX: GRADED SYMPTOM CHECKLIST <sup>9</sup> .....	23
REFERENCES.....	25



## LIST OF FIGURES

Figure 1: Pathophysiology of concussion. <sup>1</sup> .....	5
Figure 2: Return to play stages. <sup>1</sup> .....	9
Figure 3: Gross Pathology Dissections of the Four Stages of CTE. <sup>10</sup> .....	11

## **CHAPTER ONE: INTRODUCTION**

Over the past decade, professionals in the sports medicine field have debated the long term implications of concussions. Concussion is defined as a transient alteration of the brain structure caused by a direct or indirect force.<sup>1</sup> Concussion is also known as mild traumatic brain injury (mTBI).<sup>2</sup> The most common symptoms reported are headache, confusion, and disorientation.<sup>3</sup> Other signs and symptoms are nausea, dizziness, photophobia, drowsiness, seizures and emotional irritability. It is important to understand that loss of consciousness is not required to diagnose a concussion.<sup>4</sup> Sport-related concussions are often correlated to disorientation instead of loss of consciousness. According to the Center for Disease Control and Prevention, approximately 1.6 to 3.8 million Americans suffer a sport related concussion each year.<sup>1</sup> Concussions are more prevalent in sports with high speed contact such as football, hockey, boxing and lacrosse.<sup>1</sup> About 80% or more of the athletes who sustain a concussion recover within 7 to 10 days.<sup>3</sup> However, research shows that athletes who sustain a concussion are three to six times more likely to sustain a second concussion during the same season.<sup>1</sup>

During the last decade, a vast amount of clinical research on the long term effects of repetitive concussions has occurred, especially regarding chronic traumatic encephalopathy (CTE). Chronic traumatic encephalopathy is a slowly progressive neurodegenerative disease that occurs as a result of repeated head injuries such as concussions.<sup>5</sup> CTE has become a popular subject in mass media due to the increased

number of suicides and neurodegenerative diseases suffered by high profile collegiate and NFL football athletes. Boston University created a center in 2008 that specializes in researching chronic traumatic encephalopathy. They have currently collected 100 brains. Of the 19 brains obtained from deceased former NFL players, 18 out of 19 showed signs of CTE.<sup>6</sup>

According to the research, athletes who have sustained repetitive concussions are 17% more likely to develop CTE.<sup>7</sup> In other words, it is important to understand the short-term effect of concussion as well as the return to play decisions, but most importantly educate physicians, athletic trainers, coaches and athletes of the potential long-term effects of repetitive concussions. The literature on sport-related concussion has mainly focused on how to improve the return-to-play decisions rather than the potential long-term effects of sport-related concussions. The purpose of this literature review is to examine the literature pertaining to multiple concussion and the long-term effects of multiple concussion such as neurodegenerative diseases and psychological.

## **CHAPTER TWO: METHODS**

A literature review was conducted using an electronic search of the following databases: MEDLINE, Cochrane Database of Systematic Reviews, and SportDiscus. The key search terms included were concussion, and “sport concussion” and “sports concussion”. The key search terms also included depression, dementia, mild cognitive impairment, “chronic traumatic encephalopathy” (CTE), and “psychological disorder”. Additional inclusion criteria also included studies that targeted the adult athlete population who had sustained more than one concussion. Studies only were included if they were peer-reviewed, in the English language, and were published after 1990. The included studies examined the long term effects of repetitive concussion.

## CHAPTER THREE: LITERATURE REVIEW

### CONCUSSION

Concussion is a transient alteration within the brain due to a sudden physical impact. Approximately 1.6 to 3.8 million Americans suffer a sport related concussion. Concussions are caused by neuronal shearing forces that arise from rotational or angular acceleration forces to the brain. The brain damage is located at the glutamate neurotransmitter junction. Concussions happen due to the stretching of axons and disruption of neuronal membranes. When a concussion occurs, there is a state of hypermetabolism. An excitatory neurotransmitter imbalance occurs at the neurotransmitter junction (Figure 1). This imbalance causes an ionic shift, which leads to the accumulation of extracellular potassium ( $K^+$ ) and intracellular calcium ( $Ca^{2+}$ ) ions. This accumulation causes neuronal excitation and depolarization, leading to a homeostasis process where a glucose ion-pump is utilized to generate more energy (adenosine triphosphate - ATP).<sup>1</sup> Also, intracellular magnesium levels decrease, which compromises the generation of ATP, and the initiation of protein synthesis, which inhibits homeostasis of the cell membrane.<sup>8</sup>

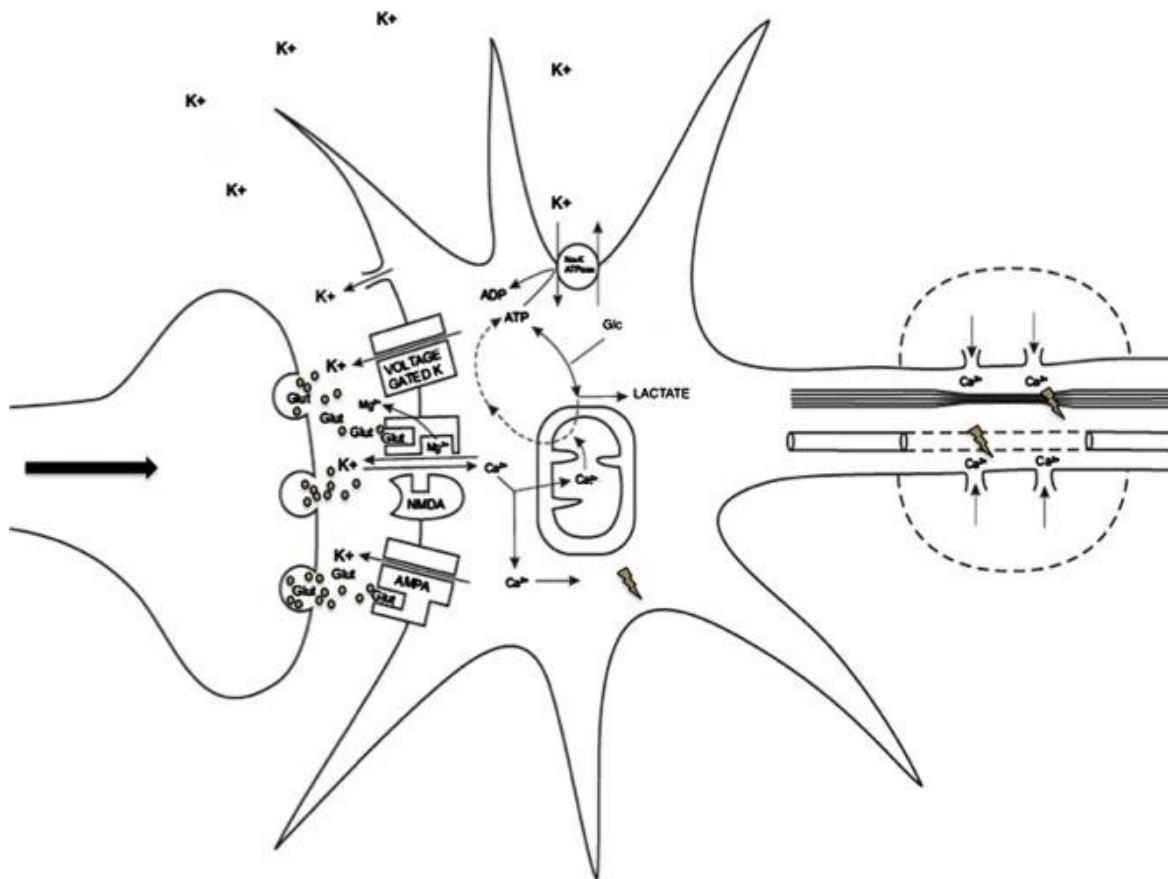


Figure 1: Pathophysiology of concussion. <sup>1</sup>

The ongoing influx of calcium ( $\text{Ca}^{2+}$ ) ions causes mitochondrial dysfunction. This dysfunction causes ATP production to decrease and subsequently, anaerobic pathways are over-utilized.<sup>8</sup> The elevation of intracellular calcium ( $\text{Ca}^{2+}$ ) also causes swelling and disruption of the axons. This is caused by metabolic and structural changes in the axons which can lead to apoptosis. Apoptosis is known as the process when a cell initiates a programmed cell death initiated. The amount of cell disruption depends on the severity of the injury. <sup>1,8</sup> Even though concussions can cause ultra-structural changes in the brain, these changes are not large enough to be visible on

neuroimaging such as MRI and CT scan. This is important to know because physicians cannot always rely on neuroimaging to tell them what is happening inside the brain.<sup>1</sup>

The common sign and symptoms of a concussion are headache, irritability, nausea, dizziness, imbalance, photophobia, fatigue, tinnitus, difficulty concentrating, mood changes, unclear thinking, and sleeping problems. More severe signs are loss of consciousness, drowsiness, disorientation, retrograde or anterograde amnesia, seizure, slow reaction time and slow verbal response.<sup>1</sup> These signs and symptoms are key clues for healthcare providers to identify. Athletes with multiple concussions demonstrate slowed recovery process compared to athletes with one concussion. As well as 92% of recurrent concussions occurs within 10 days of the initial concussion.<sup>9</sup> It is especially important that an athletic trainer be aware of them since most of the time they are the first responder at the scene. Quickly deteriorating symptoms is a sign of severe head trauma and should be recognized as a medical emergency.<sup>9</sup>

Since 1990, multiple changes have been made by sport governing bodies in an attempt to prevent concussions. These changes have allowed sports such as football to become safer and prevent the amount of fatalities due to head trauma. These changes include equipment regulation, rule changes, coach and athlete education regarding the rule changes, limitations on hours of contact practice, and athlete education on short-term and long-term of concussions.<sup>9</sup>

An on-field evaluation is required as soon as one suspects a concussion. The primary on-field evaluation should consist of assessing the airway, breathing and

circulation and ruling out any cervical spine injuries. Once other life threatening or urgent conditions have been ruled out, concussion testing can be performed. A symptoms checklist (Appendix 1) should be used and a neurological examination of cranial nerve and cerebellar function should be performed. A thorough evaluation should consist of a thorough history, observation (including, but not limited to, facial trauma and swelling), palpation of all structures of the head and neck, and special tests (Romberg test, anterograde/retrograde amnesia assessment and cranial nerve assessment, balance and pupil reaction). Performing a thorough evaluation will allow the physician or athletic trainer to determine whether a concussion has occurred. Once the athlete has been diagnosed with a concussion, he/she is no longer allowed to return to play until a series of tests are performed.<sup>1,9</sup>

Most scales used to grade concussions have been eliminated by the medical community due to the unreliability of the criteria. Many past grading scales have relied on loss of consciousness and amnesia as strict criteria. Recent research has demonstrated that it is possible to receive a concussion and not have loss of consciousness and/or amnesia, making these grading scales unreliable. In the last decade, neuropsychological testing has become the “gold standard” for evaluating concussion severity. Neuropsychological testing consists of baseline testing, which is mostly performed in pre-season (prior to any risk of concussions) and indicates the normal state of the athlete. The baseline testing components are daily symptoms, cognitive and ability assessment. Neuropsychological testing mainly focuses on short-term cognitive domains such as verbal memory, visual memory, reaction time,



information processing speed, impulse control, continuous learning and symbol scanning. Even though neuropsychological testing is highly used for sport concussion, it is very important to understand that it should not be used as a diagnostic tool for concussion, instead it should be used to measure brain recovery once a concussion has been diagnosed. For example, event-related brain potentials (ERPs) and electroencephalography (EEG) testing can identify certain areas of cognitive dysfunction after long period of time compared to the standard neuropsychological test such as ImPACT.<sup>2</sup> Neuropsychological testing can be very reliable, but there are certain factors that can limit an athlete's performance. These factors are; previous concussions, distractions, educational background, pre-cognitive functioning, cultural background, age, learning disability, language deficit, sleep deprivation, test anxiety, medications, psychiatric and hyperactivity disorders, and previous neuropsychological testing. Many of the tests performed with these limitations result in an invalid test, but there are always a few that still pass. Due to these limitations, it is suggested that further research should be performed to determine if neuropsychological testing is really the "gold-standard" testing for determining and tracking brain recovery time. It is very important to understand that there is no definitive tool used to diagnosing concussions.<sup>9</sup>

Concussion treatment varies from individual to individual. The main key points to treating a concussion are; manage and monitor symptoms, rest, reduce technology use, hydrate, and eat a proper diet. Once their symptoms have completely resolved, a return to play decision can be considered.<sup>1</sup> Return to play decisions should only be considered once the athlete is completely symptom free and has normal

neuropsychological test results, normal neurological examination results, and normal postural-stability test results. Once every test has been performed and passed symptom free the athlete should be put through a series of exertion tests prior to returning to play (figure 2).<sup>1,9</sup>

Rehabilitation stage	Functional exercise at each stage of rehabilitation	Objective of each stage
1. No activity	Complete physical and cognitive rest	Recovery
2. Light aerobic exercise	Walking, swimming or stationary cycling keeping <70% maximum predicted heart rate. No resistance training	Increase heart rate intensity
3. Sport-specific	Skating drills in ice hockey, running drills in soccer. No head impact activities	Add movement
4. Non-contact training drills	Progression to more complex training drills; for example passing drills in football and ice hockey. May start progressive resistance training	Exercise, coordination and cognitive load
5. Full contact practice	Following medical clearance participate in normal training activities	Restore confidence and assess functional skills by coaching staff
6. Return to play	Normal game play	

Figure 2: Return to play stages.<sup>1</sup>

## CTE

Chronic traumatic encephalopathy (CTE), previously known as "punch drunk" or dementia pugilistica, is a slowly progressive neurodegenerative disease caused by repeated head injuries such as concussions.<sup>5,7</sup> "Punch drunk" and dementia pugilistica

was originally used as a layman's term in the 1970s before any specific clinical evidence of CTE had emerged. Today's date chronic traumatic encephalopathy (CTE) is known as the medical term for the neuropathological consequences of repetitive concussions. CTE usually appears with insidious onset in an athlete's midlife. The mean age for CTE is 42.8 years and ranges between 25 to 76 years. CTE normally appears approximately 8 years after retirement lasts 20 years in boxers and 6 years in football players before it can results in dementia, or even a suicide attempt.<sup>5</sup>

The signs and symptoms of CTE allow classification in four stages (Figure 3). Stage one consists of confusion, dizziness, headaches and deterioration of memory and concentration. Stage two consists of behavioral changes such as irritability, anger, short-term memory loss and depression (which can lead to suicide). Stage three consists of cognitive dysfunction. Stage four of consists of diminished muscular movements, speech and visual functioning deterioration, Parkinsonism and abnormal gait and eventually dementia.<sup>10</sup> Many athletes are diagnosed with clinical dementia 10 to 30 after retirement.<sup>11</sup> De Beaumont et. al.<sup>12</sup> studied subjects who had sustained a concussion more than 30 years prior to retirement and those with no previous history of concussions. The results showed decreased performance in memory and response inhibition, as well as delayed bradykinesia (movement velocity).<sup>12</sup> All of these are

symptoms of CTE.



Figure 3: Gross Pathology Dissections of the Four Stages of CTE.<sup>10</sup>

Repetitive concussions can lead to CTE due to the increase in hyperphosphorylated tau abnormalities as well as the axonal homeostasis disturbance and loss. Concussions can cause tau proteins to become separated from microtubules in the axons due to the influx of  $\text{Ca}^{2+}$  in the neurotransmitter junction, and excitotoxicity of the glutamate transmitters. Also protein kinase and phosphatases activation leading to hyperphosphorylated tau proteins.<sup>10</sup> CTE is a progressive tauopathy. Tauopathy is a neurodegenerative disorder caused by the deposition of an intracellular accumulation of tau proteins in the brain. Tau proteins are expressed in the central nervous system and are responsible for microtubular polymerization and stability in the axons.<sup>5,7</sup> Tau proteins are regulated by protein kinase and phosphatases.<sup>1</sup> CTE is the only neurodegenerative disorder to have a specific mechanism of injury; head trauma.<sup>5,7</sup> As explained in the previous section, when a concussion occurs there is an imbalance of the signal cascade in the axons causing a disruption in homeostasis leading to

hyperpolarization and an increase accumulation of neurofibrillary tangles (NFTs), neuropil threads (NTs) and glial tangles (GTs).<sup>1</sup>

Currently, CTE has no neuropathologic diagnostic criteria. This makes it difficult to clinically diagnose an athlete with CTE. Normally athletes are diagnosed with CTE after death and an autopsy of the brain has been performed. Differential diagnoses for CTE include Alzheimer's disease or frontotemporal dementia, and Parkinson's disease. Even though CTE cannot be clinically diagnosed, there are certain biomarkers that can contribute to CTE; for example, changes in the white-matter integrity can be detected by diffusion tensor magnetic resonance imaging. As mentioned previously, CTE is caused by excessive levels of tau and phospho-tau proteins, the levels of these proteins can be measured through the extraction of the cerebrospinal fluid. These specific biomarkers allow physician to better differentiate CTE from Alzheimer's disease and frontotemporal dementia <sup>5</sup>

Gross pathology dissections of a brain with CTE (figure 3) show decreased brain weight; anterior cavum septi pellucidi with posterior fenestrations; enlargement of the lateral and third ventricle; thinning of the corpus callosum and hypothalamic floor; and atrophy of the temporal lobe, frontal lobe, parietal lobe, cerebrum, and hippocampus. Microscopic pathology dissections of CTE brains reveal neuronal loss and gliosis in the hippocampus and cerebral cortex. The dissections also showed the main characteristics of CTE; neurofibrillary inclusion in an irregular distribution such as neurofibrillary tangles (NFTs), neuropil threads (NTs) and glial tangles (GTs) within the neocortex, basal ganglia, brainstem nuclei and diencephalon. Protein tau is the main protein for

composing neurofibrillary tangles (NFTs). Brains that display signs of CTE often contain accumulation of hyperphosphorylated tau proteins. In the microscopic dissections of individual with CTE, tau proteins were commonly found in the superficial cortical laminae (layer II and III) as well as an irregular pattern through the foci in the frontal, temporal, insular cortices and white matter. These are distinct biomarkers that distinguish CTE from other tauopathies or neurodegenerative disease such as Alzheimer's disease.<sup>1,5,7</sup>

## **DEPRESSION**

Depression is defined as a psychological disorder marked by feelings of helplessness, sadness, guilt, and misery. Approximately 180,000 out of 1.6 to 3.8 million Americans who have suffered a concussion are reported to be more likely to suffer a bout of depression late in life. Females have been found to report clinical depression more often than males.<sup>13</sup>

Concussions are shown to disrupt the chemical balance within the brain, making athletes with recurrent concussion more susceptible to depression. Previous studies suggest that there are increased extracellular serotonin levels in the prefrontal and orbitofrontal cortex of people who have sustained a traumatic brain injury.<sup>14,15</sup> Research indicates that increased levels of serotonin can lead to biomechanical changes in the receptors in the extracellular region, causing depression symptoms in athletes.<sup>15</sup> Repetitive concussions can increase the excitatory neurotransmitters which could lead

to loss of neurons and cell death, putting the athletes at an even greater risk of repetitive depression episodes.<sup>14</sup>

The most common signs and symptoms that impede with daily life activities are fatigue, sadness, loss of interest, indecisiveness, sleep problems and irritability.<sup>3,14</sup> The many of the signs and symptoms of depression overlap with concussion symptoms, making it harder to diagnose the athlete with depression.<sup>13</sup> According to research, people who have sustained recurrent concussions seem to have an increased risk of developing neurodegenerative and psychiatric disorders such as clinical depression.<sup>16</sup> Furthermore, the likelihood of clinical depression seems to increase as the number of past concussions increases. Studies suggest that athletes who have sustained one or two concussions are 1.5 times more likely to be diagnosed with clinical depression than athletes with no history of concussions. Athletes who sustained three or four concussions are 3 times more likely to be diagnosed with clinical depression as they get older. According to research, 11.1% of retired athletes reported a diagnosis of clinical depression.<sup>1</sup> The prevalence of clinical depression is very serious because depression correlates to an increased rate of suicide. Clinical studies on individuals with a past history of concussions showed a risk of suicide of 18% higher and a risk of suicidal thoughts of 22% higher than the normal population.<sup>15</sup>

## **MILD COGNITIVE IMPAIRMENT (DEMENTIA)**

Dementia is defined as a neurodegenerative disorder which causes a loss of brain function. It affects brain processes such as memory, thinking, language, judgment

and behavior.<sup>17</sup> According to research, dementia in athletes who suffered recurrent concussions may not show until 10 to 30 years after the concussive incidents.<sup>11</sup> Athletes who sustained multiple concussions are shown to have a 5-fold prevalence of mild cognitive impairment.<sup>11,12</sup>

Neuroimaging shows atrophy of the hippocampal region in athletes who had multiple concussions. The hippocampal region controls memory processes. Literature suggests that an increase of the gene ApoE  $\epsilon$ 4 allele in the neuronal body causes a larger cognitive impairment and decrease neurocognitive performance after a concussion or multiple concussions.<sup>3,16</sup> An increase of ApoE  $\epsilon$ 4 in the brain has been linked to a higher risk of dementia and a decrease in the recovery time after concussions.<sup>16</sup> Apo E gene's main functions are the preservation of the neuronal structural integrity and recovery after a disturbance to the neurons and lipid transportation. Apo E gene encodes for 3 alleles ( $\epsilon$ 2,  $\epsilon$ 3,  $\epsilon$ 4). ApoE  $\epsilon$ 3 stimulates neurite growth, but ApoE  $\epsilon$ 4 inhibits neurite growth. This explains why individual with repetitive concussions has an increased number of APO  $\epsilon$ 4 gene.<sup>18</sup> Apo  $\epsilon$ 4 allele binds to tau proteins, allowing tau proteins to become hyperphosphorylated, preventing binding to microtubules, leading to a disturbance in microtubule polymerization and stability of the axons. This cascade eventually leads to the productions of NFTs.<sup>18</sup>

The most common signs and symptoms reported are impairment of memory, concentration, information processing and judgment. One cause of dementia is repetitive concussions.<sup>14</sup> Athletes who sustained repetitive concussions can display a



reduction of synaptic density and loss of neurons. These are symptoms correlated to dementia.<sup>16</sup> Athletes who had prolonged CTE can often be clinically misdiagnosed with dementia due to the high correlation between the signs and symptoms.<sup>7</sup>

## **CHAPTER FOUR: DISCUSSION**

Despite the increase of new research during the last decade, the long-term effects of concussion are still not fully known. However, research has suggested a strong correlation between the number of concussions sustained and the long-term effects such as CTE, depression, and dementia. Most studies make recommendations for further research because data is inconclusive. The literature on sport-related concussion has mainly focused on how to improve the return-to-play decisions rather than the potential long-term effects of sport-related concussions.<sup>12</sup> However, what research has been done demonstrates that repetitive concussion can lead to permanent structural and functional changes in the brain causing CTE, depression and dementia.<sup>1</sup>

### **THE ROLES OF THE ATHLETIC TRAINER**

The following sections explain the steps athletic trainers should take as first responders, educators and advocates.

#### **First Provider of Care**

Athletic trainers are commonly the first responder in an athletic medical emergency, and for this reason, they should understand the steps below:

- Athletic trainers are the front line medical response to sport concussions.

According to the NATA, athletic trainers standard of practice requires that

athletic trainers to be knowledgeable with current practice guidelines on prevention, diagnosis, treatment and return to play decisions.<sup>9</sup>

- Is very important to understand that there is no definitive tool used to diagnosing concussions. Athletic trainers have used many diagnostic tools to diagnose and measure the severity of concussions but none of these tool are 100% effective, making concussion diagnostic criteria harder.<sup>9</sup>
  - Athletic trainers must understand that concussion causes ultra-structural changes in the brain and that these changes are not large enough to be visible on neuroimaging such as an MRI or CT scan.<sup>1</sup>
- Once a concussion has been identified, the athletic training standard of care requires the understanding of concussion severity, withholding from play, and return to play decisions. Athletes who sustain a concussion are three to six times more likely to sustain a second concussion. As an athletic trainer, physician, or other medical professional, understanding this statistics and previous history of concussions is very important information to decrease the likelihood of concussions.<sup>1</sup> Athletes should not be returned to play until they are symptom free and pass a battery of tests used to ensure that their brain has recovered.
  - Tools such as event-related brain potentials (ERPs) and electroencephalography (EEG) are considered to be the “gold standard” to examine cognitive dysfunction. EEG is used to

examine electrical activity in abnormal and normal brain function.

While ERPs examine the neuroelectrical activation such as perception, memory, and action.<sup>2</sup>

- Neuropsychological testing has many limiting factors that could lead to invalid testing.<sup>9</sup>
- These different methods are important to know because athletes who have sustained multiple concussions 10 years prior to retirement are still prone to having cognitive dysfunction but passing scores on standard neuropsychological tests.<sup>2</sup>

## **Educators**

Athletic trainers are also educators to parents, athletes, coaches and medical professionals. For this reason, they should understand and follow the steps below:

- Athletic trainers can and should educate other healthcare providers, athletes, coaches and parents of the potential effects of multiple concussions.
- Athletic trainers should read and (if possible) perform research regarding concussions, have a current understanding of state and federal law, maintain best practices through the use of NATA Position Statements and medical community recommendations regarding the standard of care for

concussions. Once educated, it is imperative to implement what they know.

- Athletic trainers should educate coaches regarding the standard of care. As a coach they are encouraged to know the basic signs and symptoms of concussions and the long term implications of playing with a concussion, Coaches must also encourage athletes to be honest about their signs and symptoms.
- Athletic trainers should educate athletes and parents regarding the signs and symptoms of concussion, the severity of the injury, and the possible short-term and long-term effects.

## **Advocates**

Athletic trainers should focus on promoting awareness of the potential effects of multiple concussions by adhering to the steps below:

- Advocacy by athletic trainers, through research and education, has reduced the fatality rate from brain injuries. Changes in football (rule changes, eliminating the 3-point stance, decreasing full-contact practices, enforcing rules that help to decrease the chance of concussions, and providing better equipment such as shock absorbing helmets<sup>5</sup>) as well as changes in the prevention, diagnosis, treatment, and return to play have drastically reduced the number of brain injury related deaths in football.

The average fatality rate has dropped from 36 to 5 brain-related fatalities per year in the last decade.<sup>9</sup> Continued advocacies will reduce this number even further.

- Athletic trainers should promote compliance and knowledge of the implementation of rules aimed at preventing and treating concussions.
- Athletic trainers should advocate for licensure in all states to ensure the best quality healthcare providers possible.
- Athletic trainers should advocate for research that leads to additional rules, laws, equipment, diagnostic criteria, return to play criteria, outcomes, and follow-up research on what happens to athletes after they are concussed.

## **Limitations and Further Research**

This review of literature was limited to studies conducted on adults with previous history of concussions and signs and symptoms of CTE, depression and dementia. It was mainly limited because of amount of research that focuses on the long-term effects of concussion, leading to inclusive data. Follow up research is limited because it is difficult to follow up due to underreported and minimizing symptoms, retired and dead people, and the need for dissection to determine the diagnosis. A series of smaller, more specifically focused studies should be performed on concussions leading to long-term effects. Further research should be performed on neuropsychological testing and

the effectiveness of measuring recovery time on athletes with a history of multiple concussions as well as the criteria for invalid test.

## **APPENDIX: GRADED SYMPTOM CHECKLIST<sup>9</sup>**



<b>Graded Symptom Checklist (GSC)</b>					
<b>Symptom</b>	<b>Time of injury</b>	<b>2-3 Hours postinjury</b>	<b>24 Hours postinjury</b>	<b>48 Hours postinjury</b>	<b>72 Hours postinjury</b>
Blurred vision					
Dizziness					
Drowsiness					
Excess sleep					
Easily distracted					
Fatigue					
Feel "in a fog"					
Feel "slowed down"					
Headache					
Inappropriate emotions					
Irritability					
Loss of consciousness					
Loss of orientation					
Memory problems					
Nausea					
Nervousness					
Personality change					
Poor balance/coordination					
Poor concentration					
Ringing in ears					
Sadness					
Seeing stars					
Sensitivity to light					
Sensitivity to noise					
Sleep disturbance					
Vacant stare/glassy eyed					
Vomiting					

NOTE: The GSC should be used not only for the initial evaluation but for each subsequent follow-up assessment until all signs and symptoms have cleared at rest and during physical exertion. In lieu of simply checking each symptom present, the ATC can ask the athlete to grade or score the severity of the symptom on a scale of 0-6, where 0=not present, 1=mild, 3=moderate, and 6=most severe.

## REFERENCES

1. Khurana V, Kaye A. An overview of concussion in sport. *Journal Of Clinical Neuroscience: Official Journal Of The Neurosurgical Society Of Australasia*. 2012;19(1):1-11.
2. Broglio S, Moore R, Hillman C. A history of sport-related concussion on event-related brain potential correlates of cognition. *Int J Psychophysiol: Official Journal Of The International Organization Of Psychophysiology*. 2011;82(1):16-23.
3. Webbe F, Barth J. Short-term and long-term outcome of athletic closed head injuries. *Clin Sports Med*. 2003;22(3):577-592.
4. Clark D. Effects of repeated mild head impacts in contact sports: Adding it up. *Neurology*. 2012;78(22):e140-e142.
5. Gavett B, Stern R, McKee A. Chronic Traumatic Encephalopathy: A potential late effect of sport-related concussive and subconcussive head trauma. *Clin Sports Med*. 2011;30(1):179-188.
6. Case Studies. Center for the Study of Traumatic Encephalopathy. Available at: <http://www.bu.edu/cste/case-studies/>. Accessed November 1, 2012.
7. McKee A, Cantu R, Stern R, et al. Chronic traumatic encephalopathy in athletes: progressive tauopathy after repetitive head injury. *J Neuropathol Exp Neurol*. 2009;68(7):709-735.

8. Iverson GL. Sport-Related Concussion. In: Schoenberg MR, Scott JG, eds. *The Little Black Book of Neuropsychology: A Syndrome-Based Approach*. 2011
9. Guskiewicz K, Bruce S, Valovich McLeod T, et al. National Athletic Trainers' Association Position Statement: Management of Sport-Related Concussion. *J. Athl. Train.* 2004;39(3):280-297.
10. Woodward T, Best T. The painful shoulder: part II. Acute and chronic disorders. *Am Fam Physician.* 1, 2000;61(11):3291-3300.
11. Reeves R, Panguluri R. Neuropsychiatric complications of traumatic brain injury. *J Psychosoc Nurs Ment Health Serv.* 2011;49(3):42-50.
12. De Beaumont L, Théoret H, Lassonde M, et al. Brain function decline in healthy retired athletes who sustained their last sports concussion in early adulthood. *Brain: A Journal Of Neurology.* 2009;132(Pt 3):695-708.
13. Kontos A, Covassin T, Elbin R, Parker T. Depression and neurocognitive performance after concussion among male and female high school and collegiate athletes. *Arch Phys Med Rehabil.* 2012;93(10):1751-1756.
14. Guskiewicz K, Marshall S, Bailes J. Recurrent concussion and risk of depression in retired professional football players. *Med Sci Sports Exerc.* 2007;39(6):903-909

15. Simpson G, Tate R. Suicidality in people surviving a traumatic brain injury: prevalence, risk factors and implications for clinical management. *Brain Injury*. 2007;21(13-14):1335-1351.
16. Guskiewicz K, Marshall S, Jordan B, et al. Association between recurrent concussion and late-life cognitive impairment in retired professional football players. *Neurosurgery*. 2005;57(4):719-726.
17. Dementia. PubMed Health Web site. Available at: <http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0001748/>. Updated September 26, 2011. Accessed November 1, 2012.
18. Terrell T, Bostick R, Ewing T, et al. APOE, APOE promoter, and tau genotypes and risk for concussion in college athletes. *Clin J Sport Med: Official Journal Of The Canadian Academy Of Sport Medicine*. 2008;18(1):10-17.
19. Guskiewicz K, McCrea M, Kelly J, et al. Cumulative effects associated with recurrent concussion in collegiate football players: the NCAA concussion study. *JAMA*. 19, 2003;290(19):2549-2555.